

Electrocution Induced Cataracts and Macular Hole

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INITIAL PRESENTATION

Chief Complaint: *Objects warped inward with a blurry spot in the right eye*

History of Present Illness

The patient is a 32-year-old male with a history of myopia. Three months ago, the patient was cutting down trees at work when an electrical wire was accidentally cut leading to electrocution. He sustained diffuse burns that affected 15% total body surface area including the face, scalp, left upper extremity, left lateral trunk, and left lower trunk. Initial ocular examination around the time of the incident revealed superficial, bilateral periocular electrical burns with bilateral central corneal epithelial defects. Serial intraocular pressures were normal and there was no evidence of lens pathology. Corneal defects improved with topical erythromycin and artificial tears.

The patient now presents to clinic with continued distortion near the center of the vision of his right eye with objects appearing to be “warped inward”. He endorses similar but more mild symptoms in the left eye. He also reports a gray and blurry spot in the central visual field of the right eye extending temporally. This started approximately 2 weeks after the electrocution event when he was able to start wearing glasses again. More recently, he developed hazy vision over the entire visual field of the right eye, which is different than what he has previously been experiencing. This is significantly affecting his daily activities. He endorses rare flashes of light of unknown laterality. He denies diplopia, discomfort, redness, tearing, or floaters.

Past Ocular History

- Myopia

Past Medical History

- Accidental electrocution
- 3rd degree burns on the head
- Quadriplegia
- Scalp cellulitis and bone necrosis status post craniectomy

Medications

- Topical bacitracin
- Refresh tears
- Cyclobenzaprine
- Gabapentin
- Hydroxyzine
- Propranolol
- Trazadone

Allergies

- No known drug allergies

Family History

- Non-contributory

Social History

- Construction worker

Review of Systems

- Negative except for what is detailed in the history of present illness.

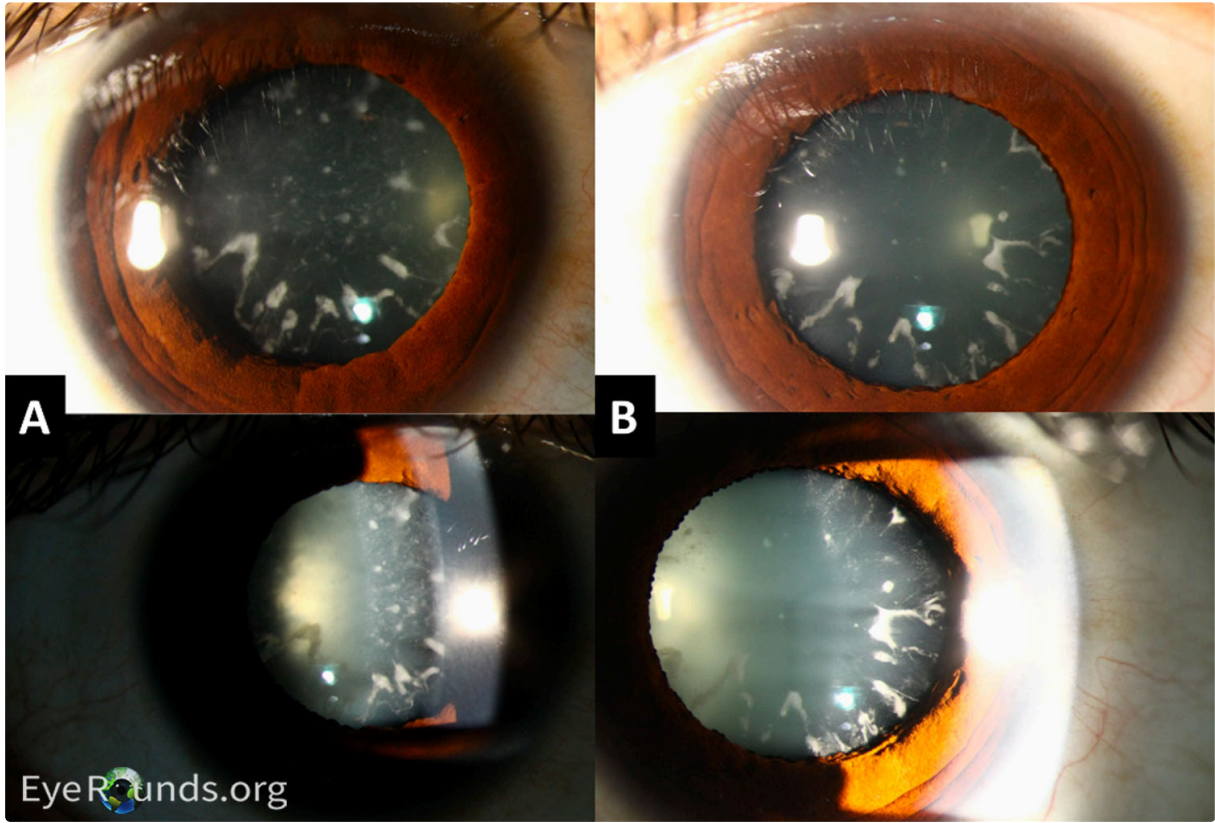
OCULAR EXAMINATION

- **Visual Acuity without correction (Snellen)**
 - Right eye (OD): 20/100ecc -1
 - Left eye (OS): 20/50 -1
- **Other Visual Acuity Tests**
 - Pinhole
 - OD: No improvement
 - OS: 20/40 +2
 - Glare Testing
 - OD: Worse than 20/400
 - OS: 20/400
- **Ocular Motility/Alignment**
 - Full OU
- **Intraocular Pressure**
 - OD: 12 mmHg
 - OS: 12 mmHg
- **Confrontation visual fields (Counting fingers)**
 - OD: Full
 - OS: Full
- **External**
 - OD: Tonic right-sided forehead contracture
 - OS: Tonic left-sided forehead contracture with scarring over the left lateral neck
- **Slit lamp examination**
 - Lids/lashes:
 - OD: Mild inferior periocular erythema, no lagophthalmos
 - OS: No lagophthalmos
 - Conjunctiva/sclera: Clear and quiet OU
 - Cornea:
 - OD: Clear
 - OS: rare punctate epithelial erosions (PEE)
 - Anterior chamber: Deep and quiet OU
 - Iris: Normal architecture and no synechiae OU
 - Lens:
 - OD: 2-3+ circumferential anterior cortical cataract, 3-4+ posterior subcapsular cataract, dulled red reflex ([Figure 1](#))
 - OS 2-3+ circumferential anterior cortical cataract ([Figure 1](#))
- **Dilated fundus examination (DFE)**
 - Vitreous: OD: Limited view, OS: normal
 - Disc: OD: Limited view with no frank pallor or edema, OS: No pallor or edema
 - Cup-to-disc ratio: OD: poor view, OS: 0.5
 - Macula: OD: poor view, OS: macular hole
 - Vessels: OD: grossly normal with limited view, OS: normal
 - Periphery: OD: grossly normal with limited view, OS: normal
- **Additional testing**
 - OCT ([Figure 2](#)):
 - OD: near full-thickness macular hole with persistent overlying ILM

- OS: central foveal hyper-reflectivity and mild disruption of all layers

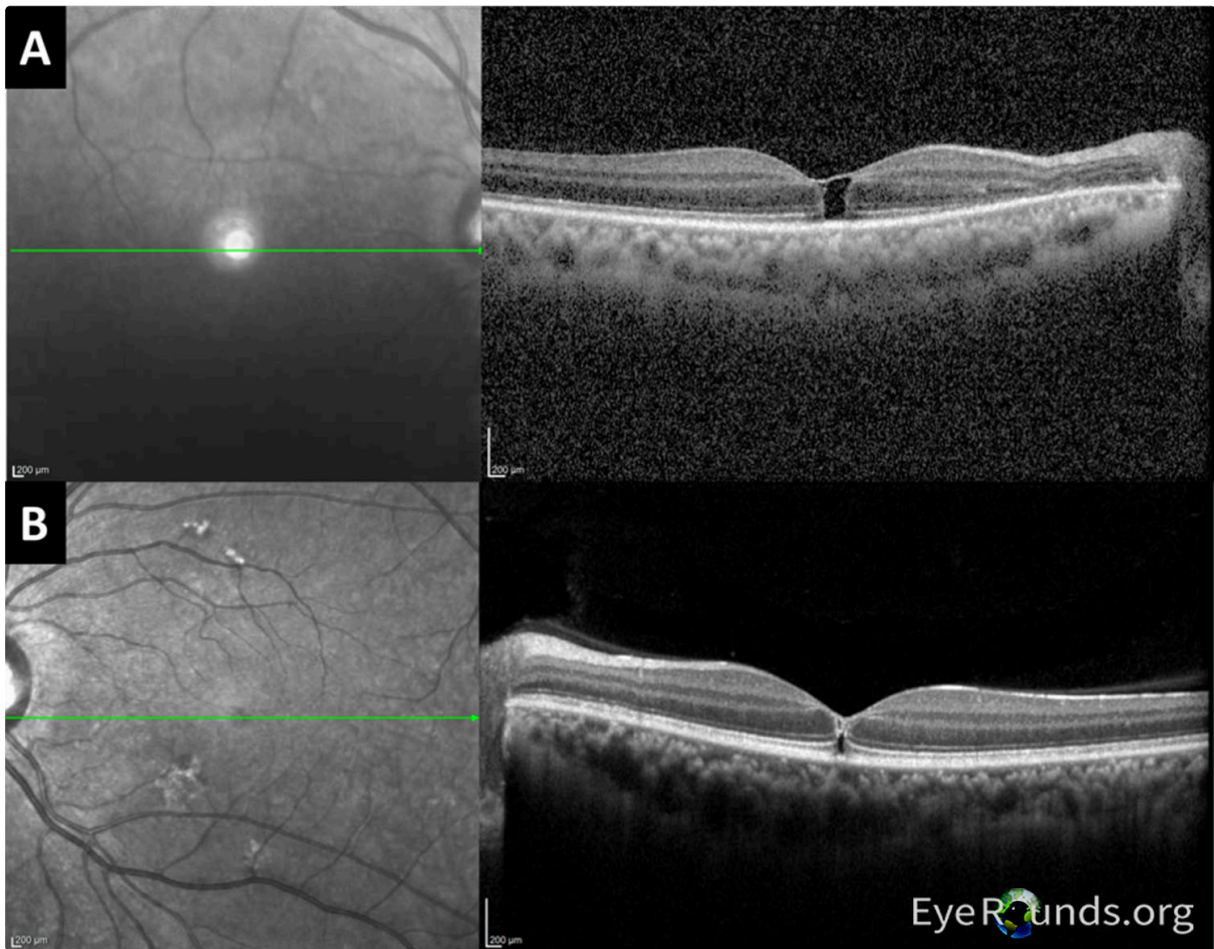
- **Differential Diagnosis**

- Electrocutation induced cataract
- Cataract from other causes
- [Full thickness macular hole](#)
- Lamellar macular hole
- Macular pseudohole



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Figure 1: External right (a) and left eye (b) photographs demonstrating bilateral cataracts following an accidental electrocution event.



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Figure 2: (A) Right eye OCT demonstrating an impending macular hole with persistent overlaying inner limiting membrane. (B) Left eye OCT demonstrating a small foveal disruption of all retinal layer.

CLINICAL COURSE

A review of the history showed a thorough eye exam near the time of admission following accidental electrocution with no evidence of prior cataracts. This information along with the rapid cataract progression over the following 2-3 months was consistent with cataract development secondary to electrocution. Subsequent bilateral phacoemulsification with intraocular lens (IOL) implantation was performed. At follow-up 4 months later, he was noted to have 20/25 visual acuity in the right eye with 20/20 visual acuity in the left eye.

The patient's complaints of warped vision, visual field loss not mapping to a neurologic region, and OCT findings were significant for a full thickness left macular hole, and the patient was referred for vitreoretinal surgical evaluation. A membrane peel surgery on the right eye was planned to follow 1 month after cataract surgery; however, the patient was lost to follow up. The patient returned 6 months later, and the left macular hole had resolved, and the right hole was noted to be resolving.

DIAGNOSIS: Bilateral Cortical Cataracts And Macular Holes Secondary To Accidental Electrocution

DISCUSSION

Etiology/Pathophysiology

A significant number of electrical injuries go unreported making electrocution estimates difficult. However, it is estimated that each year in the United States there are 4,400 cases of electric shock with around 400 deaths. (1) There are numerous reports in the literature of ocular damage following accident electrocution, typically following industrial accidents or lightning strikes. As such, a large variety of ocular sequelae following electrocution has been described. A few examples include hyphema, uveitis, anisocoria, Horner's syndrome, corneal epithelial lesions, cataract, dislocated lens, vitreous hemorrhage, retinal detachment, macular hole, central retinal vein occlusion, and optic disc edema. (2) However, due to tissue characteristics (3), the lens is the most commonly damaged structure with cataract formation. The incidence of cataract development following electrocution is estimated to be between 0.7 to 8.0%(4,5) and is most commonly anterior subcapsular (6). Reports of concurrent cataract and macular hole development are rarer. (7-12)

Electrocution events involving the head are uncommon. (5) However, the closer the shock is to the eye the more likely ocular manifestations will develop; likewise, it is rare for cataracts to form following electrocution events not involving the head. (13) Typically, 200 V is held to be the cutoff for ocular damage to occur, though retinal damage has been reported at lower voltages, although rarely. (14) One case of possible iatrogenic electric cataracts secondary to electrocardioversion has also been reported (15), though no association with electroconvulsive therapy has been demonstrated (16).

Pathophysiology

Throughout the body, electricity causes damage through two main mechanisms: thermal damage and direct effects on cells including cell membrane disruption and processes similar to electroporation. (17) Tissues with higher resistance are at an increased risk for thermal injury. Within the eye, the lens has relatively high electrical resistance compared to other ocular structures such as the optic nerve and retina. Therefore, it is the most injured component in electrocution, and thus the most extensively studied. While the exact mechanism of lens damage leading to cataract formation is unknown, leading theories include effects from protein coagulation and permeability/osmotic changes. (18,19)

Due to the lower electrical resistance of the retina and optic nerve, damage to these structures (such as full thickness macular holes) is believed to be largely related to vascular ischemia (3) with decreased likelihood of thermal injury. An exception to this is the macula which has a higher amount of melanin increasing its resistance relative to other parts of the retina. (20) The increased resistance of the macula is the leading theory explaining the propensity for macular hole development following electrocution. The exact cause of other ocular damage following electrocution is less studied but is likely related to a combination of thermal injury and direct cellular effects.

Signs/Symptoms

The signs and symptoms of ocular damage secondary to electrical injuries are directly correlated to the structure that is damaged, and in most cases have the same features as a primary disorder (such as a primary macular hole). For cataract development, the earliest sign following electrocution includes a collection of fine vacuoles beneath the anterior capsule in the midperiphery of the lens. (4) Likewise, the most common type of cataract following electrocution events is anterior subcapsular. (6) Posterior subcapsular and to a lesser extent cortical cataracts have been reported, although nuclear cataracts are rarer (6) Other signs of lens damage are related to the type of cataract that develops and is evident under slit lamp examination. Patients will typically report general cataract symptoms including clouded or blurry vision, difficulty with night vision, and sensitivity to glare.

Retinal damage such as macular holes classically leads to symptoms of decreased central vision with or without metamorphopsia. (21) Signs include a positive Watzke-Allen testing along with corresponding OCT findings. Signs and symptoms of other ocular damage will depend on the structures involved.

Workup and Management

Information about electrocution related eye damage is largely from case reports and retrospective analysis. As such, there are no definitive management guidelines. For patients presenting with electrical shocks to the head and neck or endorsing ocular complaints, early assessment by an ophthalmologist is recommended. (22) In these cases, a complete eye exam including visual acuity, eye pressures, slit lamp examination, and dilated fundus exam are important. If symptoms are indicative of retinal damage, OCT is likely the next step. The preservation of eyelid structure and function is also important to help prevent secondary corneal damage due to desiccation. It should be noted that a complete eye exam can be difficult in these patients as they will likely have other systemic injuries making transportation to an eye clinic difficult.

As the patient becomes stabilized, future management will be guided by the structures injured. Cataract development is the most common finding and in approximately 80% of cases will progress and require surgical intervention with phacoemulsification and IOL implantation. (5, 24) Historically, cataract development was believed to either be immediate or delayed following a variable latency period with two to six months being the most common time until diagnosis though more than a year had also been reported. (5,13,25) A newer retrospective cohort study disputed the delayed development of cataracts following electrocution. (26) However, patients are still typically followed with frequent eye exams.

There is less information available about the management of macular holes in electrical injuries. As in this case, macular holes have been noted to spontaneously resolve following a period of watchful waiting. In one study, the majority of patients with macular holes following lightning strikes were managed conservatively. (27) If no resolution is noted following conservative management over a couple of months, surgical intervention is indicated, likely including PPV.

<p>EPIDEMIOLOGY OR ETIOLOGY</p> <ul style="list-style-type: none"> • Electrocutation related eye damage is rare with cataract formation the most common finding (0.7-8.0%) • At least 200 V is required for most ocular injuries • Ocular damage/cataract formation is more common with entry or exit site on the head 	<p>SIGNS AND SYMPTOMS</p> <ul style="list-style-type: none"> • Based on the underlying complication: <ul style="list-style-type: none"> ◦ Cataract: clouded/blurry vision, difficulty with night vision, and sensitivity to glare ◦ Macular hole: decreased central vision ± metamorphopsia
<p>PATHOPHYSIOLOGY</p> <ul style="list-style-type: none"> • The exact cause of tissue damage is unknown and is likely multifactorial: <ul style="list-style-type: none"> ◦ Thermal injury from tissue electrical resistance (lens > retina/optic nerve) ◦ Cell membrane disruption ◦ Vascular ischemia (likely more important in retinal injuries) 	<p>TREATMENT/MANAGEMENT</p> <ul style="list-style-type: none"> • Complete ophthalmologic assessment including visual acuity, intraocular pressure, slit lamp exam, dilated fundus exam ± OCT • Cataract: surgery is definitive • Macular hole: includes both watchful waiting and surgical intervention • Other treatment depends on the underlying damage

Related Link(s):

- [Mature cortical cataract \(atlas entry\).](#)
- [Incipient cortical cataract: the inferior nasal quadrant axiom \(atlas entry\).](#)
- [Cataracts: From One Medical Student to Another \(tutorial\).](#)
- [Macular Hole, Full-Thickness, Extrafoveal/Eccentric \(atlas entry\).](#)
- [Macular hole, commotio retinae, and choroidal rupture after blunt eye injury \(atlas entry\).](#)
- [Macular Hole \(atlas entry\).](#)
- [Full-Thickness Macular Hole \(case\).](#)

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